Is Smoker/Nonsmoker Segregation Effective in Reducing Passive Inhalation among Nonsmokers?

STUART JAY OLSHANSKY

Abstract: Using expired carbon monoxide (CO) and a test of coordination as measures of tobacco smoke exposure in a natural environmental setting where smokers and nonsmokers were segregated, results indicate that by comparison to a control group, subjects seated in adjacent smoking/nonsmoking environments were not only exposed to similar ambient levels of CO, but also show similar physical and physiological reactions to their exposure in the form of coordination test scores, expired CO, and blood carboxyhemoglobin. While the results may not be generalized to other tobacco smoke constituents or other environmental settings, they raise questions about the health benefits of smoker segregation which future research must address. (*Am J Public Health* 1982; 72:737–739.)

Introduction

Tobacco smoke has been known to be a human carcinogen and has been closely associated with a number of acute and chronic illnesses in the smoker.[1] Nonsmokers who breathe in the smoke of nearby smokers (passive inhalation) are exposed to tobacco smoke constituents at levels sometimes higher than those to which smokers are exposed.[2–4] There is clinical evidence to indicate that passive inhalation of tobacco smoke for extended periods of time (including the accumulated effects of repeated short exposures) may incur some health-related problems in nonsmokers.[5–12] Nevertheless, short-term low-level exposure to tobacco smoke or its hazardous constituents has not yet been shown to result in negative health consequences for nonsmokers (compare, for example, references 13–15 and 16,17).

In response to these and other clinical studies, the Environmental Protection Agency (EPA) delineated national primary air quality standards for carbon monoxide (CO) in the United States in order to protect individuals against the occurrence of blood carboxyhemoglobin (COHb) levels above two per cent.[18]* In spite of the fact that ambient levels of CO produced by the burning of tobacco exceed the federal ambient air quality standards,[19] the federal standards were not intended to affect the incidence of smoking in public places or enclosed environments.

Historically, federal, state, and local legislators throughout the United States have attempted to impose antismoking legislation designed to limit the environments in which tobacco may be burned.[20] In general, the authors of antismoking legislation have asserted that their legislation will lead to: a reduction of the annoyance that nonsmokers experience as a result of passive inhalation; and the elimination of potential health hazards for nonsmokers. While most of the proposed antismoking bills based on the latter premise have failed to pass into law,[21] some of the bills designed to reduce the annoyance factor of passive inhalation have been successfully legislated in the form of smoker/nonsmoker segregation laws.[21–28] However, segregation laws simply do not specify how far nonsmokers should be situated away from smokers in an enclosed area, nor are there operationally defined limits for exposure to the "annoying" or "dangerous" constituents of tobacco smoke.

This paper is a case study of a single environmental setting in which smokers and nonsmokers were segregated in an enclosed area. It attempts to address the question of whether or not segregation is effective in reducing objective correlates[29,30] of tobacco smoke exposure among the nonsmokers situated in a nonsmoking area.**

Materials and Methods

The location of the experimental measurements was a weekly bingo game in a Knights of Columbus Hall located in the city of South Milwaukee, Wisconsin. This 68 × 46 × 10-foot hall is centrally air conditioned with a single ceiling vent located near the center of the room and has six ceiling-mounted Honeywell electrostatic fan-operated air cleaners (Model F54c-389s). Twenty per cent of the available seating was designated for nonsmokers, and there were no smokers in the nonsmoking section during the measurements.*** The

*COHb is a blood-born product of the inhalation of CO and has been associated with health effects ranging from mild impairment of psychomotor coordination to serious illness and even death. The two per cent figure was based on evidence that low levels of COHb in human blood may be associated with impairment of ability to discriminate time intervals.[14]

Address reprint requests to Stuart Jay Olshansky, Population Research Center, University of Chicago, 1126 E. 59th Street, Chicago, IL 60637. This paper, submitted to the Journal August 24, 1981, was revised and accepted for publication December 10, 1981.

**This paper is not an inquiry into whether or not the passive inhalation of tobacco smoke is occurring among nonsmokers at levels that might be considered "annoying" or "dangerous" to their health.

*** The estimated average number of smokers at any point in time during the experimental measurements was approximately 20, or 11.1 per cent of the average occupancy of the hall during the bingo game (200) minus the number of subjects (18). This figure is based on the estimate that about one-third of US adults are smokers,[31] and one-ninth or 11.1 per cent of every group of average adults will be smoking at any one point under most social conditions.[32]

air conditioner and all six air cleaners were functioning on the high setting during the experimental measurements. Control group measurements were taken in a designated nonsmoking area of the Marquette University Memorial Library in Milwaukee, Wisconsin. Nonsmoking sections of the library consisted of entire floors, thus ensuring a smoke-free environment.

Twenty-seven paid nonsmoking subjects (ages 20–32) volunteered for the study. Subjects were blocked by sex and randomly assigned to two experimental groups and one control group.

Ambient and subject breath CO and blood COHb measurements were measured through the use of an ECOlyzer (Model-2800) and 6 × 6-inch saran bags (Anspec, Inc.) especially formulated for taking breath samples.[33–35]

Psychomotor coordination was analyzed with a standardized eight-minute coordination test (Flanagan Aptitude Classification Test, Coordination; No. 7-3426-Science Research Associates, Inc.).‡

The two experimental groups provided pre-test breath samples in the nonsmoking floor of the library, then traveled by car to the bingo hall. Subjects assigned to the nonsmoking section sat as far from the smokers as possible (approximately 15 feet separated them from the nearest smoker). Subjects assigned to the smoking section sat in the middle of that section. Breath and ambient air samples were obtained one hour after arrival in their respective areas and at hourly intervals for two more hours. Breath sample bags were sealed and delivered to an adjacent room by an assistant, thus making this a single blind study. Control group measurements took place the same day and time the following week in the nonsmoking floor of the library. Following the final air samples, subjects took the Flanagan coordination test.

Results

According to the data in Table 1, pretest levels of ambient CO, mean expired CO, and mean blood COHb levels were comparable for the two experimental groups and the control group.

Following exposure to ambient air in the enclosed experimental environments, the subjects seated in the smoking and nonsmoking areas of the bingo hall had comparable levels of exposure to ambient CO and subsequent levels of expired CO and blood COHb throughout the three-hour testing period.

The Flanagan coordination test results showed significant differences between the mean coordination scores of the control group (76.1) and each of the experimental groups (nonsmoking-66.1, smoking-57.1; t = 2.44, α .05).‡‡ There were no significant differences in the coordination scores of the subjects in the smoking and nonsmoking environments (t = 1.56, N.S.)

TABLE 1—Ambient CO (ppm) at Time of Breath Sample, Mean Expired CO (ppm), and Mean Blood COHb (%) of Subjects in Smoking, Nonsmoking, and Control Groups

	Expired CO ($\bar{x}$ ppm)			
		Hour		
Subjects	Pre-Test (% COHb)	1 (% COHb)	2 (% COHb)	3 (% COHb)
Smoking	3.67	5.64	7.38	8.99
	(0.73)	(1.13)	(1.48)	(1.80)
Ambient CO (ppm)	2.2	10.0	9.8	11.7
Nonsmoking	3.86	5.69	7.39	9.22
	(0.77)	(1.14)	(1.48)	(1.84)
Ambient CO (ppm)	2.2	9.6	10.7	12.5
Control	3.93	3.96	3.83	3.99
	(0.79)	(0.79)	(0.77)	(0.80)
Ambient CO (ppm)	3.0	3.1	3.0	3.0

Analysis of variance shows no significant differences in pretest measurements between the three groups. An analysis-of-variance (two-factor with repeated measures) of expired CO levels for the three groups determined that there were significant increases in mean expired CO in both experimental groups as a function of the time of exposure to passive inhalation ($p < .001$; df 3,72), and that the experimental groups showed comparable and significant differences in their mean expired CO ($p < .001$; df 2,24) by comparison to the control group.

Discussion

Within this particular environmental setting, ambient levels of CO, expired CO, and blood COHb, were almost identical for subjects seated in adjacent smoking/nonsmoking environments. The most likely explanation is that the CO produced by the burning of tobacco simply diffused uniformly throughout the entire hall. In the absence of a determination of air-flow conditions and the effectiveness of the air cleaners, however, it is impossible to determine the relative contributions of either of these factors to the dispersion of CO throughout the experimental environments.

These findings may not be extended to the diffusion of other "dangerous" or "annoying" tobacco smoke constituents because no attempt was made to measure them. The Flanagan coordination test could be questioned as an "objective" measure of CO exposure in this case because of the possibility of subject sabotage, and because there is no mean coordination score below which one might attribute its cause to any single constituent of tobacco smoke. Nevertheless, the findings raise a number of questions on the benefits of segregation.

The study demonstrated that one volatilized constituent of tobacco smoke, CO, was experienced and reacted to in an identical fashion, by people seated in adjacent smoking/nonsmoking environments, thus inferring that other substances may have diffused throughout the nonsmokers envi-

‡This test is sensitive to, and has recently been associated with, blood COHb levels as a result of the inhalation of pure carbon monoxide.[36]

‡‡This represents a control/nonsmoking comparison. Only one T test was done because the mean coordination score for the smoking group was even less than that of the nonsmoking group.

ronment to the same extent. Indeed, all of the subjects in both experimental groups reported informally subjective annoyance symptoms of teary eyes, itchy nose, headache, coughing, and nasal congestion.

Only one subject in the entire study had a blood COHb level that reached two per cent after the three-hour exposure period. This is not considered a dangerous level of COHb.[14] The ambient levels of CO were also well below the federal standards for short-term exposures.[18] However, studies have shown that the accumulated effects of repeated short exposures may incur some health-related problems in nonsmokers.[8–12] Thus the question of whether segregation is always effective in protecting the nonsmoker from "dangerous" constituents of volatilized tobacco smoke can also be asked.

In summary, although these results cannot be extrapolated to other environments they raise questions regarding the benefits of smoker/nonsmoker segregation. In order to more adequately address these questions, research needs to focus on: defining the concentrations of "annoying" and "dangerous" levels of volatilized tobacco smoke constituents to which humans are particularly sensitive; determining whether the existing segregated environments sanctioned by existing laws reduce the exposure of nonsmokers to "non-annoying" and "safe" levels; and describing the types of environments and air-flow conditions that are conducive to reaching such levels.

REFERENCES

1. Smoking and Health: A Report of the Surgeon General. Washington, DC: Govt Printing Office, 1979. DHEW Publ. No. *(PHS)* 79-50066.
2. Schmeltz I, Hoffman D, Wynader EL: The influence of tobacco smoke on indoor atmospheres. Prev Med 1975; 4:66–82.
3. Hoegg UR: Cigarette smoke in closed spaces. Environ Health Perspect 1972; 2:117–128.
4. Russell MA, Cole PV, Brown E: Absorption by non-smokers of carbon monoxide from room air polluted by tobacco smoke. Lancet 1973a; 1:576–579.
5. Wald N, Howard S, Smith PG, *et al:* Association between atherosclerotic diseases and carboxyhemoglobin levels in tobacco smoke. Br Med J 1973; 1:761–765.
6. Cuddeback JE, Donovan JR, Burg WR: Occupational aspects of passive smoking. An Ind Hyg Assoc J 1976; 37:263–267.
7. Manning FA, Feyerabend C: Cigarette smoking and fetal breathing movements. Br J Obstet Gynaecol 1976; 83:262–270.
8. Colley JRT: Respiratory symptoms in children and parental smoking and phlegm production. Br Med J 1974; 2:201–204.
9. Cameron P: The health of smoker and nonsmokers' children. J Allergy Clin Immunol 1969; 43:336–341.
10. Colley JRT, Douglas JWE, Reid DD: Respiratory disease in young adults: influence of early childhood, lower respiratory track illness, social class, air pollution, and smoking. Br Med J 1973; 3:195–198.
11. Bonham GS, Wilson RW: Children's health in families with cigarette smokers. Am J Public Health 1981; 71:290–293.
12. Hirayama T: Non-smoking wives of heavy smokers have a higher risk of lung cancer: a study from Japan. Br Med J [Clin Res] 1981; 282:183–195.
13. Beard RR, Wertheim G: Behavioral impairment associated with small doses of carbon monoxide. Am J Public Health 1967; 57:2012–2022.
14. Beard RR, Grandstaff N: Carbon monoxide exposure and cerebral function. Ann NY Acad Sci 1970; 174:385–395.
15. Schulte JH: Effects of mild carbon monoxide intoxication. Arch Environ Health 1963; 7:524–530.
16. Stewart RD: The effect of carbon monoxide on humans. J Occup Med 1976; 18:304–309.
17. Stewart RD, Newton PE, Hosko MJ, *et al:* Effect of carbon monoxide on time perception. Arch Environ Health 1973; 27:155–160.
18. Environmental Protection Agency: National Primary and Secondary Ambient Air Quality Standards. Federal Register 1975; 40:50.
19. Goldsmith JR, Beard RR, Dinman BD: Epidemiological Appraisal of Carbon Monoxide Effects. The Effects of Chronic Exposure to Low Levels of Carbon Monoxide on Human Health, Behavior, and Performance. Washington, DC: National Academy of Sciences and National Academy of Engineering, 1969, p 32.
20. Cobey C: The resurgence and validity of antismoking legislation. University of California (Davis) Law Review 1974; 7:167–195.
21. State Legislation on Smoking and Health 1980. Atlanta GA: US Department of Health and Human Services, Centers for Disease Control, 1981.
22. 14 CFR Part 252 (1974) (Docket No. 21708; EDR 231).
23. Ibid, p 19147.
24. Health Aspects of Smoking in Transport Aircraft. Washington, DC: National Institute for Occupational Safety and Health, Federal Aviation Administration, US Departments of Transportation, and of Health, Education, and Welfare, 1971.
25. Smoking by Passengers and Operating Personnel on Interstate Buses 14 M.C.C. 256 (November 17, 1971). From a speech by Burns DM: Scientific Evidence on the Hazards of Involuntary Smoking. Workshop on Rights of Non-smokers, held at the University of Maryland, January 11, 1975, p 4. National Interagency Council on Smoking and Health, New York, N.Y.
26. S. 2219, H.R . 1309, H.R. 5279, 93rd Congress, 1st Session, 1973.
27. Final Rule, 44 FR 5071, January 25, 1975 (ER-1091). NPRM, 44 FR 29486, May 21, 1979 (EDR-377, Docket No. 29044) in Agenda of Significant Regulations, Civil Aeronautics Board, Washington, DC, 1980.
28. National Association of Motor Bus Owners vs United States, 370 F. supp. 408 (D.D.C. 1974), aff'g 114 M.C.C. 256 (1971).
29. Vogt TM, Selvin S, Widdowson G, *et al:* Expired air carbon monoxide and serum thiocyanate as objective measures of cigarette exposure. Am J Public Health 1977; 67:545–549.
30. Rawbone RG: Carbon monoxide in alveolar air as an index of exposure to tobacco smoke. Clin Sci Mol Med 1976; 51:495–501.
31. Department of Health, Education and Welfare: Adult use of Tobacco, 1975. Center for Disease Control, Atlanta, GA 1976.
32. Repace JL, Lowrey AH: Indoor air pollution, tobacco smoke, and public health. Science 1980; 208:464–472.
33. Bay HW, Blurton KF, Sedlak JM: Electrochemical technique for the measurement of carbon monoxide. Anal Chem 1974; 46:1837–1839.
34. Peterson JE, Stewart RD: Predicting the carboxyhemoglobin levels resulting from carbon monoxide exposure. J Appl Physiol 1975; 39:633–638.
35. Stewart RD, Stewart RS, Stamm W: Rapid estimation of carboxyhemoglobin level in firefighters. JAMA 1976; 235:390–392.
36. Stewart RD, Peterson JE, Baretta ED, *et al:* Experimental human exposure to carbon monoxide. Arch Environ Health 1970; 21:154–164.

ACKNOWLEDGMENTS

This study was performed in partial fulfillment for the master's degree in Psychology at Marquette University. Mr. Olshansky is currently working on a doctorate in Sociology at the University of Chicago. The author wishes to acknowledge the contributions made to this study by Dr. Jack Peterson, Dr. Kenneth Matyniak, D. J. Wyman, R. Tuma, Argonne National Laboratory, and the Milwaukee Jewish Federation.